Appendix F

Literature Survey of Emaciation for Great Horned Owls

REDISTRIBUTION OF ORGANOCHLORINES INTO THE LIVERS OF EMACIATED BIRDS

The literature on the redistribution of organochlorines (OCs) that occurs in birds that become emaciated, especially in situations where emaciation results from dieldrin poisoning, was studied. We did not find papers that reported laboratory studies on the redistribution of PCDD/Fs in birds, but we found four papers that are relevant to the situation faced by the BAS in interpreting the residues of PCDD/Fs in the livers of emaciated owls (presumably poisoned by dieldrin) on site. Here we summarize each of the papers individually and then make an over-all assessment.

Stickel et al. 1966

Part of this paper reports results from an experiment in which Japanese quail were exposed to various dietary concentrations of dieldrin. When half of the birds in each dietary group had died of dieldrin poisoning, the remainder were sacrificed. The sacrificed birds had been exposed, on average, for longer than the birds that died, and thus had slightly higher tissue residues. Otherwise, the birds killed by dieldrin poisoning and sacrificed groups were comparable. Thus, if allowance is made for the higher initial residues in the sacrificed group, the two groups represent birds before and after the process of hypophagia and emaciation that results in death from dieldrin poisoning. A complicating factor is that the birds were divided into three groups exposed to different dietary concentrations (10, 50, and 250 ppm dieldrin, dry weight, in the diet); the dates of death were correspondingly different (50–153 days, 9–72 days, and 5–14 days, respectively). Hence, although the total intakes and body burdens were comparable among the three groups, the schedules of exposure were different. For this analysis, Stickel et al. pooled birds from the three dose groups, recognizing that they are not strictly comparable. Information on tissue residues was presented only for males. Data from a fourth group exposed to dieldrin at 2 ppm in the diet were not used because none of the birds died.

To compare tissue residues in birds before and after the process of emaciation and death, we estimated the total body burden in each bird by multiplying the total body mass at the time of death or sacrifice (from Table 8-I in the study) by the concentration of dieldrin in the carcass remainders (from Table 8-III in the study). This requires the assumption that the average dieldrin concentration in the tissues removed from the carcass (liver, brain, beak, feet, skin, and gastrointestinal tract) were similar (or at least proportional) to those in the carcass remainders. This procedure yielded the following data for the 18 individual males for the data reported (D = died and S = sacrificed) as shown in **Table F-1**.

Table F-1. Dieldrin liver concentrations per dosage

Bird No.	Dosage (ppm)	Day of Death	Est. Body Burden (μg)	Liver, Dieldrin % lipid	Liver, Dieldrin conc (ppm)	Liver Lipids, Dieldrin (ppm)
613	250	5 D	1,033	1.57	18.0	1146
614	250	8 D	1,241	1.21	14.8	1233
615	250	9 D	2,099	1.92	16.0	838
616	250	9 D	547	1.20	25.9	2176
624	50	9 D	538	1.28	19.8	1545
625	50	20 D	1,905	1.45	29.1	2007
626	50	28 D	3,086	2.71	51.7	1915
635	10	50 D	756	1.91	5.7	300
820	10	143 D	1,804	1.05	24.0	2317
826	10	146 D	2,341	0.49	-----	-----
618	250	11 S	4,870	7.70	48.7	633
619	250	11 S	1,535	5.03	15.0	300
627	50	30 S	5,825	3.32	36.5	1099
628	50	30 S	990	21.31	140.8	661
629	50	30 S	5,109	14.27	81.1	569
824	10	158 S	4,213	18.33	93.5	510
825	10	158 S	2,687	2.39	6.1	256
822	10	158 S	1,640	0.82	2.7	329

The geometric mean residues in the two groups are shown in **Table F-2**.

Table F-2. Geometric mean residues of liver dieldrin concentrations

	Carcass Liver Burden (μg)	Liver % lipid	Liver, Dieldrin conc. (ppm)	Liver lipids, Dieldrin conc. (ppm)
Died (N = 10)	920	1.65	19.7	1301
Sacrificed (N = 8)	1,650	9.14	28.8	491
Ratio (S/D)	1.79	5.54	1.46	0.38

Thus, the geometric mean body burden in the sacrificed birds was 1.79 times higher than that in the birds that died. This reflects the fact that they were exposed for longer than the birds that died within each dose group (third column in Table F-1). We make the assumption that the liver residues would have been smaller in the same ratio (1:1.79) at the times when the birds died in the same dose groups. Accordingly, we estimate the geometric mean concentration of dieldrin in the livers would have been about 16.1 ppm (28.8/1.79) at the time when the birds died in the same dose groups. Hence, the best estimate of the fraction by which liver concentrations increased during the process of dieldrin-induced starvation and death is 19.7/16.1, or a 1.2-fold increase. The reason why this factor is so small is that the percentage of lipid in the liver decreased by about the same factor (5.5) as the percentage of lipid in the carcass remainders (5.4).

This estimate is uncertain because of the assumptions that had to be made about differences in exposure, the pooling of data from the three dose groups, and the wide variability in tissue residues among individuals, even within dose groups (see **Table F-1**).

Robinson et al. 1967

In this study, domestic pigeons and Japanese quail were experimentally poisoned with dieldrin. The quail study provided no data on liver residue concentrations that can be used to help address the emaciation issue at RMA. There were seven groups of dosed groups of pigeons, but six of these groups provided no information of use to the RMA issue with owls because (i) single doses were used, with consequently uninterpretable pharmacokinetics, and (ii) all doses killed all birds. Thus, comparisons of residue levels between live and dead birds was not possible.

The most relevant component of this study is the group of pigeons exposed to 50 ppm dieldrin in the diet (n = 7). For birds that died (n = 3) due to dieldrin exposure, the mean time to death was 90 days, and the geometric mean concentration of dieldrin in liver at death was 62.1 ppm (Table 4 in the study). For comparison, the birds that survived (n = 4) had a mean concentration of 22.9 ppm in the liver (Table 5 in the study). The birds that died had been exposed for only half as long (90 days versus 180 days) to dieldrin. Therefore, the concentration of dieldrin in their livers at the onset of dieldrin-induced starvation would have been between 0.5 and 1.0 times that in the survivors at 180 days, depending on the pharmacokinetics (1.0 if steady state had been attained at 90 days, 0.5 if the liver concentrations increased linearly from 90 days to 180 days). Therefore, the study suggests that dieldrin concentration increased by a factor between 2.7 (i.e., 62.1 ppm/22.9 ppm) and 5.4 (62.1 ppm/11.45 ppm).

Apart from the difference in exposure periods (and consequent uncertainty about the pre-starvation level in the liver), another important limitation of this study is the small sample sizes (n = 4, 3). Because the concentrations were widely variable within groups (ranging from 11.8 to 51.2 ppm in the four survivors), this leads to large statistical uncertainty in the derived ratios.

Ecobichon and Saschenbrecker 1969

DDT was administered to 5-week-old White Leghorn cockerels at three dietary concentrations (250 μg/day, 500 μg/day, and 1000 μg/day) for three time periods (15 weeks, 10 weeks, and 5 weeks, respectively). Each group was then divided into two sub-groups. One sub-group (n = 10 birds per dose level) received a normal ration of untreated diet, the other group (n = 10 birds per dose level) received a restricted (50% of normal food consumption) untreated diet.

The food-deprived birds fed the 1000 μg DDT/day (Group 3) and the 500 μg DDT/day (Group 2) responded very quickly to the effects of food restriction. Birds in Group 3 were all dead within 10 days; birds in Group 2 were all dead by Day 13. In contrast, food-deprived birds in Group 1 required a long time to respond, with one bird living 48 days after the initiation of the restricted diet. Despite the variability in time to death between and within groups, direct comparison of the food-deprived birds with their corresponding control groups is valid because one control bird was sacrificed on the same day that each food-deprived bird died.

Data summarized in Figures 1, 2, and 3 of the paper allow the following conclusions regarding the effect of food-deprivation on liver concentrations of DDT/DDE are shown in **Table F-3**.

Table F-3. Effect of food-deprivation on liver concentrations of DDT/DDE

Sample Group	Concentration of μg DDT/day	Approximate liver concentration of DDT/DDE
1	250	**4-fold increase**
2	500	**1.5-fold decrease**
3	1000	**1.8-fold increase**

De Freitas and Norstrom 1974

This was a pharmacokinetic study in which domestic pigeons were exposed to PCBs (Aroclor 1,2,5,4) and then subjected to various treatments which led to redistribution of residues within and among the body tissues. PCBs were administered in gelatin capsules at doses of about 8 mg/kg/day for 11 days. The most relevant comparison is between the "after-dosing" group (N = 6), which were fed uncontaminated food for 3 days post-dosing and then killed, and the "stressed" group (N = 6), that were fed for 3 days post-dosing, starved for 7 days in the cold, and then killed. Geometric mean PCB concentrations in the liver were estimated as 16 ppm in the "after-dosing" group and 64 ppm in the "stressed" group (Table 6 in the study), indicating a 4-fold increase during starvation. PCB concentrations were estimated in this study using "peak 15." This is only a minor component in Aroclor 1254, but was used as a reference because there was no evidence that it was metabolized. Hence, the ratio between the estimated PCB concentrations in the liver of the two groups should be a good estimate of the ratio of the concentrations of "peak 15," but not necessarily good estimates for other components of the mixture. Peak 15 was thought to be primarily a hexachlorobiphenyl. The main limitations of this study are (i) although starved, the birds did not die from starvation; and (ii) the exposure period was only 11 days. The exposure was insufficient to reach an equilibrium distribution in the tissue, especially for a hexachlorobiphenyl.

Overview

The four studies summarized above give a range of estimates of the degree to which concentrations of OCs in the livers of birds change during food-deprivation including dieldrin-induced starvation. These estimates range from a decrease of 1.5- fold to an increase ranging from 1.2- to 5.4-fold. However, it must be emphasized that all the studies had important limitations. The most important of these were (i) the small sample sizes (3–10 per dose group, largest in the study by Ecobichon and Saschenbrecker); (ii) the fact that none of the studies involved exposure to PCDD/Fs, but all involved exposure to other OCs; and (iii) the fact that none of the studies involved exposure of owls, taxonomically related species, or ecologically similar species. Nevertheless, we are impressed by the fact that all the studies yielded quantitatively similar results and that none of them suggested a large increase in OC concentrations in the liver during starvation. The conventional wisdom is that starvation should lead to mobilization of OCs and consequently to increases in the tissue concentrations of OCs. However, the data reviewed in this paper suggest that liver lipids are depleted at about the same rate as lipids in other tissues. Hence, mobilization of OCs during starvation apparently does not always lead to substantial increase in wet-weight concentrations of OCs in liver tissues.

For these reasons, we propose that it should be assumed that wet-weight concentrations of PCDD/Fs in the livers of great horned owls could remain about the same or increase up to 5-fold during the process of hypophagia and starvation that accompanies dieldrin poisoning. Therefore, the concentrations of dieldrin in the livers of dieldrin-poisoned owls could be divided by a factor from 1 to 5 for comparison with those of non-poisoned owls.

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